

REMARKS:

Claims 6, 32-33 and 35-37 were rejected under 35 USC 102(a) as being anticipated by Maine.

It is respectfully requested that the examiner reconsider this rejection in view of the inventor's comments in the attached affidavit. Specifically, as noted in paragraph 5, Mayne et al used XeC to protect neurons from cell death caused by a specific HIV protein (tat). As discussed in paragraph 4, neurons are not susceptible to HIV infection. As such Mayne cannot provide any teachings or conclusions concerning the use of XeC to treat an HIV infection. The differences are summarized best in paragraph 5 wherein it is stated that 'Mayne et al proposed XeC as a way of protecting neurons from cellular suicide caused by calcium release while our data ... show[s] that XeC can induce cellular suicide in PBMCs infected with HIV'.

Claims 6, 32-33 and 35-37 were rejected under 35 USC 103 as unpatentable over Petit et al and Stingl et al, in view of DeBarieri et al.

It is respectfully requested that the examiner reconsider this rejection in view of the inventor's comments in the attached affidavit. As discussed therein, while structurally related, the actions of XeC and XeD and XeE have not been shown to be equivalent. As further discussed therein, only XeC demonstrated specific inhibition of IP3-mediated calcium release. Furthermore, as discussed in the inventor's affidavit, not all retroviruses behave in a similar manner and not all retroviruses require NF-kB. As such, one skilled in the art, on consideration of the cited references might conclude that it would be 'worth a try' to determine if XeD and XeE could be used to treat other retroviral diseases, but there would be no guarantee of success. Furthermore, while structurally related, XeC has different activities than XeD and XeE and as such these references do not teach or suggest that XeC could be used to treat any retroviral disease.

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Further and more favorable consideration is respectfully requested.

Respectfully submitted

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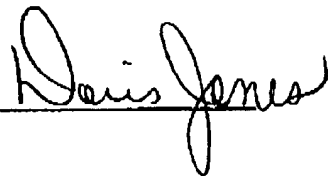
November 1, 2006

Enc.()

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